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ABSTRACT

This paper summarizes recent research on functions of the cochlea of the inner ear. The cochlea is described as the seat of the first step in the auditory sound analysis and transduction of mechanical vibration into electrochemical processes leading to the generation of neural action potentials. The cochlea is also described as a frequent seat of auditory disorders. This research summary addresses findings concerning: the classical model of cochlear functioning developed by ter Kuile and modified by H. Davis; explanations for the sharp tuning and great sensitivity of the auditory nerve fibers and inner hair cells; the role of the outer hair cells in cochlear acoustic emissions; contradictions of the classical model of hair cell stimulation posed by evidence for the depolarization of the hair cells during basilar membrane displacement; effects of removing the outer hair cells; possible mechanisms for hair cell depolarization; increased damping of the cochlea at high sound intensities; and the nature of the cochlear pitch code. Figures and graphs illustrate these findings. Contains 49 references. (DB)

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Chapter 3

COCHLEAR PROCESSES: A RESEARCH REPORT

JOZEF J. ZWISLOCKI

Institute for Sensory Research
Syracuse University

The cochlea of the inner ear is an organ of great interest to scientists, audiologists, and otologists. As you well know, the cochlea is the seat of the first step in the auditory sound analysis and of transduction of mechanical vibration into electrochemical processes that lead to the generation of neural action potentials. The cochlea is also a frequent seat of auditory disorders. Accurate diagnosis of these disorders and their eventual treatment or prevention depend on our understanding of cochlear mechanisms.

In the past two decades, a prodigious number of discoveries has revolutionized our concepts of cochlear function. These concepts are only partially included in even the most recent textbooks and handbooks, and in no place is an integrated picture of the cochlear function presented. I will attempt to present it here and to contrast it with the views we held before the revolution took place.

First, let me recall some functional anatomy to which I will be referring. Figure 1 shows in cross section the second turn of the cochlea of a Mongolian gerbil, our main experimental animal. The structures that can be seen are quite similar to corresponding human structures. I am sure you can recognize the basilar membrane with the organ of Corti and the tectorial membrane. The latter is displaced somewhat toward the spiral limbus and away from the organ of Corti due to histological artifact. The normal position of the tectorial membrane is parallel to the reticular lamina of the organ of Corti. It is possible to identify the outer hair cells by their somewhat darker staining and an inner hair cell on the inner side of the tunnel of Corti. It is well established now that the stereocilia of the outer hair cells are firmly coupled to the tectorial membrane and that the coupling of the stereocilia of the inner hair cells is less tight.

More than 40 years ago Békésy (1947) was able to show that sound was propagated in the cochlea in the form of transversal traveling waves on the basilar membrane, whose energy was dissipated before they reached the helicotrema in the cochlear apex so that no wave reflection took place. This discovery is still valid. According to Békésy's observations, the waves produced a local vibration maximum as they ran along the cochlea, and the location of the maximum changed with sound frequency, moving from cochlear base toward its apex as sound frequency decreased. The dependence of the location on sound frequency resembled the relationship between the subjective pitch and sound frequency, and Békésy assumed that the location constituted the adequate code for pitch, as had been first suggested by Helmholtz in the mid-19th century. The cochlear waves Békésy drew according to his observations are reproduced in Figure 2 for two instants in time. The waves clearly exhibit a vibration maximum, but the maximum is rather flat, and Békésy assumed that it was sharpened up in the nervous system to account for the fine frequency resolution our hearing is capable of. The cochlear frequency resolution, as seen by Békésy (1947) in human postmortem preparation is shown for several cochlear locations in Figure 3. At every location, the vibration amplitude is drawn as a function of frequency. The dashed lines show the results of my mathematical theory (Zwislocki, 1946, 1948, 1950), which provided the physical foundation for Békésy's waves. The agreement of the theoretical results with the experimental ones suggests that the mechanism of Békésy's waves was sufficiently well understood. The theory also accounted well for the empirical location of the vibration maximum along the cochlea.

Since the 19th century, it was believed that vibration of the basilar membrane led to excitation of the hair cells, and at the onset of the 20th century, ter Kuile (1909) provided an explicit model of hair cell stimulation. Because the outer hair cells are located near the middle of the basilar membrane, where the amplitude of vibration must be the greatest, and the inner hair cells are located near its edge, where the amplitude must be small, it was believed that the outer hair cells transmitted the auditory information near the threshold of audibility and the inner hair cells transmitted at higher sound intensities (Stevens & Davis, 1938).

The model of ter Kuile was modified by H. Davis (1958) without changing its principle. It is schematized in Figure 4. According to this model, the hair cell excitation was produced by deflection of the hair cell hairs, or stereocilia, resulting from shear motion between the tectorial membrane and the reticular lamina. The shear motion was due to the geometry of the basilar membrane and the organ of Corti and of the tectorial membrane. It was assumed that,

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FIGURE 1. Second turn of a Mongolian gerbil cochlea in cross section. The various anatomical parts should be familiar, but note the somewhat more darkly stained outer and inner hair cells and the somewhat displaced tectorial membrane because of histological artifact. Undisturbed, the undersurface of the tectorial membrane is parallel and in close proximity to the reticular lamina.

as seen in the cross section, the basilar membrane and the tectorial membrane motions could be approximated by rotation of two stiff beams around two mutually offset axes. As illustrated in Figure 4, such motions must lead to stereocilia deflection toward the cochlear modiolus during basilar membrane displacement in the direction of scala tympani, and toward the outer wall during basilar membrane displacement in the direction of scala vestibuli. It was found later on that, in the first instance, the cells were hyperpolarized, and in the second, depolarized (Flock, 1971, for review). The latter was associated with the excitation of auditory nerve fibers. The displacement of the basilar membrane toward scala vestibuli, then, was assumed to produce neural excitation.

In the early 1960s, the model of cochlear function sketched above appeared to be well established. Then, one dogma fell after another. Demonstration by Kiang (1965) and his co-workers that the auditory nerve fibers were very sharply tuned eliminated the need for central sharpening of the auditory frequency analysis. In 1967, Spoendlin discovered that almost all afferent auditory nerve fibers end on the inner hair cells, not the outer hair cells. This made the assumption that the outer hair cells coded sounds at low intensities quite improbable and strongly suggested that the sharp tuning and great sensitivity found by Kiang's group referred to fibers ending on the inner hair cells. It also made the role of the outer hair cells enigmatic. In the same year, Johnstone and Boyle (1967) showed that the local vibration maximum of the basilar membrane is much sharper in live guinea pigs than had been observed by Békésy on postmortem preparations. Rhode (1971, 1973) confirmed these results on live monkeys and demonstrated directly that death was at least a partial cause of the decreased sharpness of tuning in Békésy's experiments. The effect of death on basilar membrane tuning found by Rhode is schematized in Figure 5. A few years later, Russell and Sellick (1977) discovered through pioneering intracellular recordings that the inner hair cells are as sharply tuned as

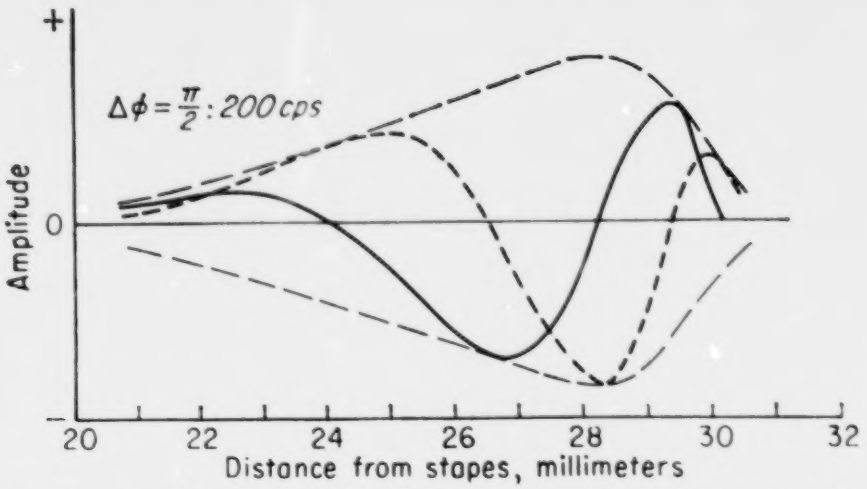

FIGURE 2. Traveling cochlear waves drawn by Békésy for two instants of time according to his stroboscopic observations. Note that the wave length becomes shorter with the distance from the stapes and the amplitude goes through a local maximum. (Békésy, 1947, as reproduced in Zwislocki, 1950).

the nerve fibers. This means that the sharp tuning found in the nerve fibers is of cochlear origin. The question whether the tuning of the inner hair cells is sharper than that of the basilar membrane has remained controversial. Nevertheless, research at the Massachusetts Institute of Technology (MIT) (Frishkopf et al., 1982; Holton & Weiss, 1983; Peake & Ling, 1980) on lizards has shown that the hair cells can be sharply tuned in the absence of any basilar membrane tuning.

Two additional key discoveries provided an explanation for the increased sharpness of cochlear tuning in a live cochlea. Kemp (1978) discovered that the cochlea was capable of emitting sound, especially when excited by a sound impulse. Five years later, Brownell (1983) found that the outer hair cells vibrate longitudinally when exposed to an alternating electrical field. Since the outer hair cells generate such a field in the cochlea in the form of cochlear microphonics, conditions for a positive electromechanical feedback are provided. It is known in the engineering world that positive feedback can lead to spontaneous oscillation, and it is believed that the electromechanical oscillation of the outer hair cells is the cause of the cochlear acoustic emissions. It is also known that a positive feedback counteracts damping and sharpens frequency resolution. Its existence in the cochleas of live animals but not postmortem can explain why cochlear tuning becomes less sharp after death. It also clarifies the role of the outer hair cells which, by providing a positive electromechanical feedback, appear to serve as a kind of cochlear amplifier (Davis, 1983). When the outer hair cells are damaged or missing, the cochlear sensitivity decreases, and a hearing loss results, even when the inner hair cells appear undamaged (e.g., Dallos & Harris, 1978; Liberman & Kiang, 1978; Schmiedt et al., 1980).

For a feedback to be positive, it must be in phase with the input signal to the system. Several attempts have been

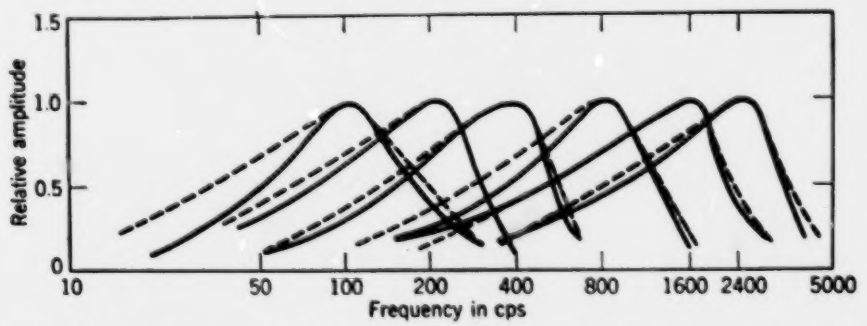

FIGURE 3. Cochlear filter functions determined by Békésy at various locations along the cochlear spiral (solid lines) and calculated according to my original theory (dashed lines). Note that the filter functions, determined postmortem, are not very sharp. (Zwislocki, 1950).

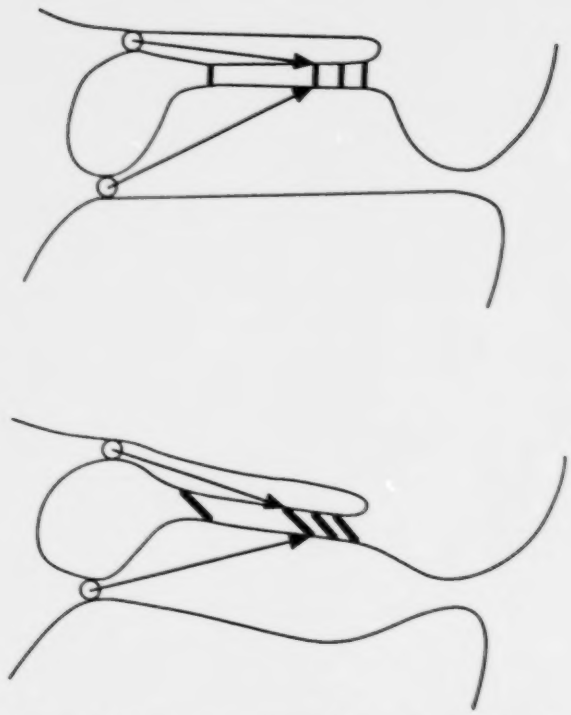

FIGURE 4. Schematic representation of the shear motion between the tectorial membrane and the reticular lamina according to the classical model. In the upper panel the basilar membrane is in its zero position; in the lower panel, it is displaced toward scala tympani. In association with this displacement, the hair cell stereocilia are deflected toward the modiolus, a hyperpolarizing direction.

made at modelling the cochlear feedback (e.g., Neely & Kim, 1983; Mountain et al., 1983; Ashmore, 1986), but the models have not been explicit enough to define unambiguously the feedback phase. As a result, they have not brought to evidence that the classical concept of hair cell stimulation, on which they have been based, leads to a negative rather than positive feedback. This relationship can be derived from Figure 4, given the well-established fact that the outer hair cells become shorter when they are depolarized or excited. As already explained, this is associated with the stereocilia deflection toward the outer wall and, according to the classical model, with the basilar membrane displacement toward scala vestibuli and the tectorial membrane. The shortening of the outer hair cells produces a relative displacement of the reticular lamina away from the tectorial membrane, counteracting the effect of the basilar membrane displacement. As a result, the shear displacement between the tectorial membrane and the reticular lamina is reduced, and with it, the hair cell depolarization. This amounts to a negative feedback. In the lower panel of Figure 4, the basilar membrane is drawn displaced in the opposite direction, toward scala tympani, and the stereocilia deflected toward the modiolus—the hyperpolarizing direction. This is associated with a lengthening of the outer hair cells and a relative displacement of the reticular lamina toward the tectorial membrane. Again, the effect of the basilar membrane displacement away from the tectorial membrane is counteracted and the shear displacement reduced. To produce a positive feedback, the excitation phase of the outer hair cells would have to be reversed. They would have to be depolarized during basilar membrane displacement toward scala tympani. Under such conditions, the outer hair cells would be shortened, producing a relative displacement of the reticular lamina in the same direction as the displacement of the basilar membrane. This means that the shear displacement between the tectorial membrane and the reticular lamina, and with it the depolarization of the hair cells, would be enhanced—a positive feedback.

Depolarization of the hair cells during basilar membrane displacement toward scala tympani is in direct contradiction of the classical model of hair cell stimulation, and difficult to accept for many scientists. Nevertheless, experimental evidence against the model has been accumulating for almost 20 years. It started with Konishi and Nielsen's (1973) discovery made with the help of very low sound frequencies that most auditory nerve fibers become excited and increase their firing rate during basilar membrane displacement toward scala tympani. Remember that neural excitation is coupled to hair cell depolarization. Because, in their experiments the cochlea had to be opened and the possibility of an artifact was increased, the discovery was not taken seriously at first. However, it was confirmed in my laboratory without tampering with the cochlea (Zwislocki & Sokolich, 1973; Sokolich et al., 1976; Schmiedt & Zwislocki, 1980).

The experiments also showed that elimination of the

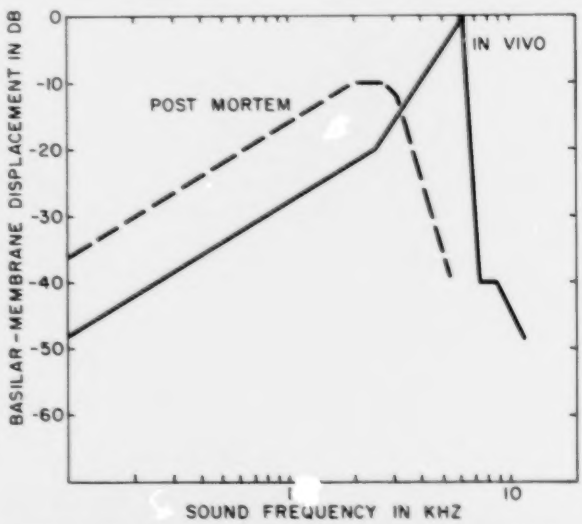

FIGURE 5. Magnitude of basilar membrane vibration as a function of sound frequency for a basal cochlear location schematized according to Rhode's results. The solid line corresponds to in vivo conditions; the intermittent one, to post mortem conditions. Note the difference in the sharpness of tuning and in the best frequency between the two conditions. (Zwislocki, 1980).

outer hair cells reversed the phase of neural excitation, bringing it in line with the classical model. In other words, in portions of the cochlea devoid of outer hair cells, the inner hair cells seemed to be stimulated according to the classical model. This was not true in the portions with a full complement of outer hair cells.

An individual example of the correlation between the neural response phase and the preservation of outer hair cells is shown in Figure 6. The solid line and open circles show the remaining outer hair cells in percent as a function of cochlear location. The intermittent line and closed circles do the same for the inner hair cells. The triangles indicate the inferred cochlear locations of innervation of nerve fibers. The closed triangles indicate neural excitation during basilar membrane displacement or motion toward scala tympani; the open ones, excitation during basilar membrane displacement or motion toward scala vestibuli, in agreement with the classical model. The correlation between the response phase and the availability of outer hair cells is striking.

Several series of neural recordings performed by others in the presence of normal cochleas essentially confirmed our results (e.g., Sellick et al., 1982; Ruggero & Rich, 1983, 1987, 1988; Ruggero et al., 1986). However, they seemed to be contradicted by direct recordings from the inner hair cells, which were consistent with the classical

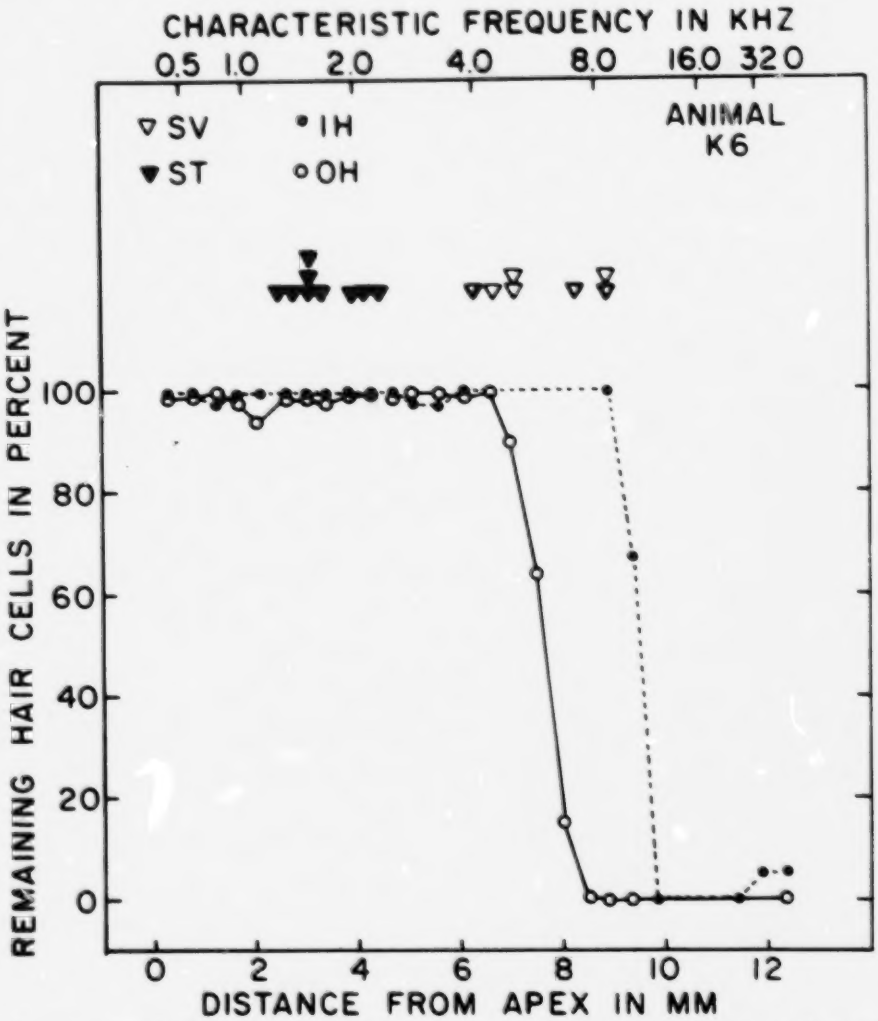

FIGURE 6. Correlation between the percentage of remaining hair cells and the response polarity of the auditory nerve fibers. The solid line shows the percentage of OHCs as a function of the distance from the apex, and the dashed line, that of the IHCs. Note the window of preserved IHCs in the presence of depleted OHCs. In this window, the fibers were excited (increased firing rate) during inferred basilar membrane displacement or motion toward scala vestibuli. In the presence of practically complete sets of both cell types, the excitation occurred during basilar membrane displacement or motion in the opposite direction—toward scala tympani. (Sokolich et al., 1976).

model (Sellick & Russell, 1980; Nuttal et al., 1981). Subsequently, it was shown that the apparent disagreement was likely to have originated, in part, from differences among animal species (Oshima & Strelioff, 1983), and in part, from an artifact produced by the electrode lodged in the organ of Corti during hair cell recordings (Zwislocki, 1984; Zwislocki & Smith, 1988a).

More recently, it became possible to measure directly the response phases of the outer hair cells. According to the obtained results, the cells are depolarized during basilar membrane displacement toward scala tympani, except at very low sound frequencies and/or very high sound pressure levels. When the sound frequency is varied from very low to high, a phase shift of about 180° can be detected. The same is true when the sound pressure level is increased beyond 80 dB (e.g., Zwislocki & Smith, 1988b; Zwislocki, 1990). These relationships are reproduced in Figure 7 for one outer hair cell. The phase is plotted as a function of log frequency by means of the sinus of the phase to avoid 180° and 360° phase ambiguities. The thinner curve was obtained at 40 dB SPL, the thicker one, at 100 dB SPL. The phase reversal is clearly apparent near the best frequency indicated by the amplitude maximum in the 40-dB curve. The phase shift decreases toward the low frequencies, at the left of the figure. A small positive bump in the thinner curve at the left and a positive slope of the thicker curve at the right are artifactual.

How is it possible for the outer hair cells, and also the inner hair cells, to be depolarized (excited) during basilar membrane displacement toward scala tympani, contrary to the plausible geometrical explanation provided by the classical model? First, it has been found that the stereocilia bundles of the outer hair cells are very stiff (e.g., Flock, 1977; Strelioff & Flock, 1984) and the tectorial membrane, very compliant (Zwislocki et al. 1988; Zwislocki & Cefaratti, 1989), contrary to the assumptions inherent in the classical model. As a consequence, the tectorial membrane cannot act as a stiff anchor for the tips of the stereocilia, but acts rather as a mass load. During basilar membrane oscillation, the mass is driven by the stereocilia, which act as stiff springs. It is well known that a mass driven through a spring is subject to a resonance effect. Near the resonance frequency, its amplitude of oscillation becomes much larger than the amplitude driving the opposite end of the spring. Projected on the cochlear structures, this means that the tectorial membrane vibrates with a larger amplitude than the reticular lamina in the shear motion direction. In the transversal direction, the tectorial membrane must vibrate with the same amplitude as the reticular lamina because of the incompressible fluid between them.

The situation can be modeled by a vibrator with a flexible reed attached nearly vertically to its armature, as shown in Figure 8 (Zwislocki, 1980). The vibrator represents a short segment of the basilar membrane; the reed, corresponding

FIGURE 8. An electrodynamic vibrator as a simple qualitative model of a short section of the basilar membrane with the organ of Corti and the tectorial membrane. The bent tube to the right points to the vibrator armature acting as the organ of Corti. The reed over the armature simulates bundles of OHC stereocilia, and a small nut at its tip, the mass of the tectorial membrane. Note that the magnitude of lateral vibration of the reed tip is much greater than the magnitude of vertical vibration of the armature. This is true only near the transversal resonance of the reed. (Zwislocki, 1980).

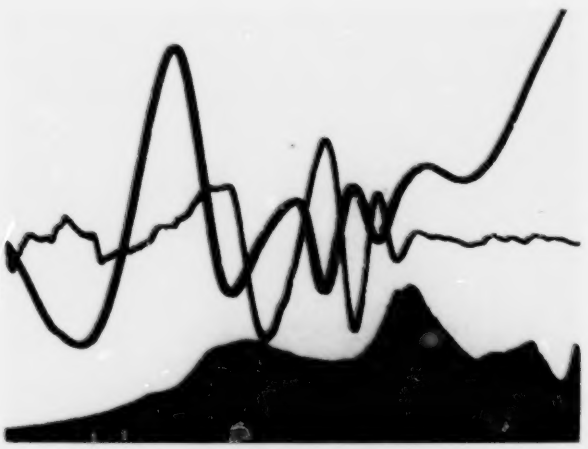

FIGURE 7. Response phases of an OHC plotted as sine functions of sound frequency. The thinner line is for a SPL of 40 dB, the thicker one, for a SPL of 100 dB. Note that in the vicinity of the best frequency (greatest magnitude of the 40 dB curve), the two curves are in phase opposition. The phase difference decreases toward lower sound frequencies (to the left; the small bump in the thinner curve is artifactual). (According to Zwislocki, 1988.)

stereocilia bundles, and a small weight at the top of the reed, represent a corresponding section of the tectorial membrane. The vibrator armature moves vertically at a small amplitude barely perceptible in the photograph taken under stroboscopic illumination. The model tectorial membrane oscillates at a right angle to the oscillation of the armature with a much larger amplitude. The relatively large amplitude is what is needed to reverse the phase of hair cell depolarization, as illustrated in Figure 9. In the upper panel, the basilar membrane with the organ of Corti and the tectorial membrane are schematized in their rest positions. The stereocilia of the three rows of outer hair cells and one row of inner hair cells extend almost vertically from the reticular lamina. In the lower two panels, the basilar membrane is displaced toward scala tympani. The middle panel is drawn for low sound frequencies, well below the resonance frequency of the stereocilia tectorial membrane system. The tectorial membrane is pulled toward the outer wall by the stiff stereocilia of the outer hair cells. Its resulting radial displacement deflects the stereocilia of the inner hair cells slightly toward the outer wall, a depolarizing direction.

The situation corresponds to neural excitation during basilar membrane displacement toward scala tympani. The stereocilia of the outer hair cells are deflected slightly toward the modiolus, a hyperpolarizing direction, because of the elastic reaction force of the tectorial membrane. The lowest panel is for sound frequencies near the tectorial membrane resonance. Here, the radial oscillation amplitude of the tectorial membrane is assumed to be larger than the corresponding oscillation amplitude of the reticular lamina, and the stereocilia of the inner hair cells as well as of the outer hair cells are deflected outward. This situation corresponds to depolarization of both the inner as well as outer hair cells. The response phases illustrated in Figure 9 are in agreement with phase measurements on the hair cells as well as on the nerve fibers. It should be pointed out, however, that the deflection direction of the stereocilia of the inner hair cells in the middle panel can be either outward, as drawn, or toward the modiolus, depending on the balance of the tectorial membrane and reticular lamina displacements at the location of the inner hair cells.

Resonance of hair cell stereocilia has been demonstrated directly in lizards (Frishkopf & De Rosier, 1983). This is not yet possible for mammalian cochleas. However, indirect evidence for the resonance of the mammalian stereocilia loaded with the tectorial membrane is strong. On the basis of stiffness measurements of the stereocilia and the mass of the tectorial membrane, Strelioff et al. (1985) calculated that the resonance would coincide approximately with the best frequency. The resonance provides the only viable explanation for the complex response phases of the hair cells exemplified in Figure 7. Together with the positive feedback, it accounts for the complicated cochlear response characteristics in the intensity and frequency domains.

Why does the response phase of the hair cells change by 180° in the vicinity of their best frequency when the sound pressure level is increased beyond 80 dB? Very likely because the positive feedback becomes ineffective (e.g.,

FIGURE 9. Schematic representation of shear motion between the tectorial membrane and the reticular lamina according to the empirically correct new model. In the upper panel, the basilar membrane is in its zero position, in the lower two panels, it is displaced toward scala tympani. The middle panel holds for low sound frequencies, the lowest panel, for the vicinity of the best frequency (resonance of the stereocilia-tectorial membrane system). In the middle panel, the OHC stereocilia are deflected toward the modiolus (inhibition), but the IHC stereocilia, toward the lateral wall (excitation). In the lowest panel, all the stereocilia are deflected toward the lateral wall (excitation).

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Kemp, 1978), and the inherent damping of the cochlea prevents the resonance of the stereocilia-tectorial-membrane system. Under such conditions, the relative motion between the tectorial membrane and the reticular lamina follows the classical model.

The increased damping at high sound intensities also affects the response amplitude of the hair cells. This is illustrated in Figure 10, which shows the alternating receptor potential of an outer hair cell as a function of sound frequency plotted logarithmically on the horizontal axis. Every curve has been obtained at a different sound pressure level in 10-dB steps. The lowest curve in the bottom panel corresponds to 20 dB, and the highest, to 50 dB. In the upper panel, the sound pressure level increases from 50 to 80 dB. The amplification was decreased by a factor of 6 for the upper panel. If the system were linear, with constant damping, the response amplitude would increase linearly. This means that the amplitudes of each curve would be about three times greater than the amplitudes of the curve immediately below it, in agreement with the 10-dB steps in sound pressure level. Clearly, the amplitude ratios are much smaller, especially at high sound pressure levels.

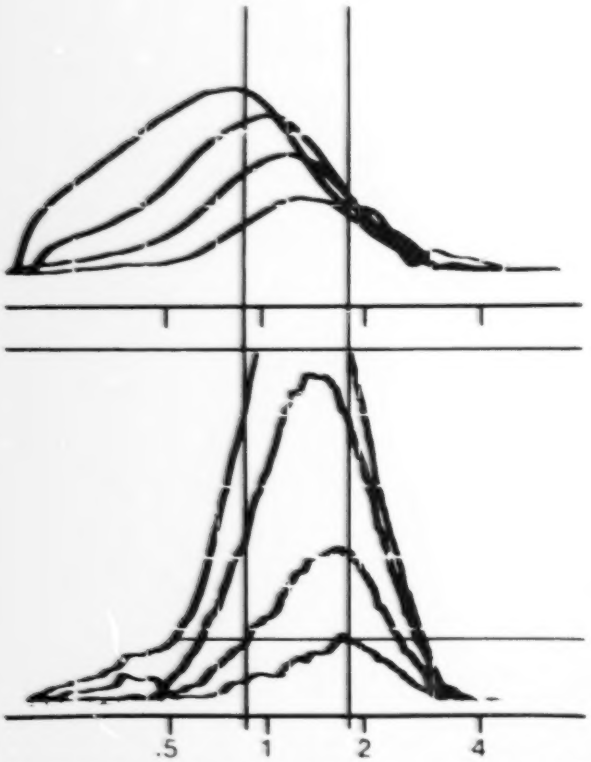

FIGURE 10. Transfer functions (magnitude vs. sound frequency) of an OHC with SPL as a parameter (20–50 dB in the lower panel, 50–80 dB in the upper panel). The amplification in the upper panel is reduced by a factor of 6. The frequency scale is logarithmic, the magnitude scale, linear. Note the compression of the magnitudes at the higher SPLs and the shift of the maximum toward lower frequencies. Note also the bunching of the curves at the high frequency cutoff. (Zwislocki, 1991.)

In addition to the amplitude compression evident in Figure 10, the response maximum is gradually shifted to lower sound frequencies as the sound pressure level is increased. Between 20 and 80 dB, the shift amounts to roughly one octave (Zwislocki, 1991). It has been believed for over a century that the location of the cochlear response maximum determined the subjective pitch. But the relationship between the pitch and sound frequency changes very little with sound intensity (e.g., Stevens & Davis, 1938). How then can the response maximum whose relationship to sound frequency changes by one octave within an intensity span of 60 dB be the adequate code for pitch? According to Figure 10, only the high-frequency cutoff of the response curves remains approximately independent of sound intensity. Could it constitute the cochlear pitch code?

With this latest insight, I will end my list of cochlear concepts that have undergone radical changes since 1965. I think you will agree with me that a revolution in our understanding of the cochlear function has taken place.

ACKNOWLEDGMENTS

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